Original Article

MEDICINE PALLIATIVE CARE

DOI: 10.47582/jompac.1347667

*J Med Palliat Care*. 2023;4(5):423-430

# Evaluation of laboratory and radiological imaging results in terms of hospitalization and mortality in acute pancreatitis cases

**Burak Akın, Burak Demirci, Abuzer Coşkun**

Department of Emergency Medicine, İstanbul Bağcılar Training and Research Hospital, İstanbul, Turkey

**Cite this article as**: Akın B, Demirci B, Coşkun A. Evaluation of laboratory and radiological imaging results in terms of hospitalization and mortality in acute pancreatitis cases. *J Med Palliat Care*. 2023;4(5):423-430.

**Received**: 22.08.2023 ◆ **Accepted**: 16.09.2023 ◆ **Published**: 27.10.2023

## ABSTRACT

**Aims**: Acute pancreatitis (AP) is a common diagnosis in emergency services and is very important in terms of morbidity and mortality. It was aimed to evaluate the relationship of laboratory and imaging findings of AP cases with hospitalization and mortality outcomes.

**Methods**: This retrospective study was conducted with 225 cases over the age of 18 who applied to the emergency department between 1 September 2020 and 1 March 2021. Age, gender, laboratory and imaging data of the patients were recorded and evaluated on groups formed according to hospitalization and mortality status.

**Results**: The mean age of 225 acute pancreatitis cases was 54.10±19.07 years, and 116 (51.6%) were female. 169 (75.1%) individuals were hospitalized and seven (3.1%) patients resulted in mortality. Age was associated with mortality ($p<0.001$). Lipase levels in the mortality group were substantially higher, with 3474.71±3013.69 U/L ($p= 0.046$). Similarly, elevated urea was found to be related with mortality ($p=0.019$). On ultrasonography, pancreatic edema was found to be associated with mortality ($p=0.012$). The presence of intrahapatic bill duct dilatation ($p=0.002$), pancreatic edema ($p=0.045$) and peripancreatic fluid ($p=0.009$) in magnetic resonance cholangiopancreatography (MRCP) was significant at hospitalization. Tomography and MRCP findings did not correlate with mortality.

**Conclusion**: Laboratory parameters and imaging findings in the emergency department may be predictive of hospitalization and mortality outcomes in AP.

**Keywords**: Acute pancreatitis, emergency department, laboratory, imaging methods, hospitalization, mortality

## INTRODUCTION

Acute pancreatitis (AP) is a prevalent gastrointestinal disorder with a high global incidence, frequently leading to emergency department visits and hospital admissions.[1] According to recent research, the prevalence of acute pancreatitis (AP) ranges from 4.9 to 73.4 instances per 100,000 individuals globally.[2] Its annual cost can reach 2.6 billion dollars and it is observed quite widely.[3] Although the mortality for AP in the general population remains constant, its incidence is increasing due to diagnostic methods and ease of admission.[2] There is currently no established pharmacological intervention that has been empirically validated. AP is a pathological condition that can be clinically detected in the absence of systemic manifestations. This condition can lead to both local and systemic inflammatory responses, organ dysfunction, pancreatic necrosis, and ultimately, mortality.[4]

While gallstones and alcohol are the etiology of 80% of AP cases, the remaining are less common causes such as drug reactions, pancreatic solid and cystic malignancies, and hypertriglyceridemia.[5] The diagnosis of AP necessitates the presence of prototypical abdominal pain, high levels of serum amylase and/or lipase that exceed three times the upper limit of normal, and/or the identification of at least two diagnostic abnormalities on abdominal imaging.[6,7] Patients commonly experience pain in the central epigastric region and the upper right quadrant. The sensation of discomfort has the potential to extend towards the posterior region or laterally. The object in question may possess characteristics reminiscent to a belt, with the knife being affixed in a manner that allows it to remain in a fixed position. In order to establish a diagnosis for the disease, it is necessary to consider the collective evaluation of anamnesis, physical examination, laboratory tests, and radiographic investigations.[8,9] In emergency medical care, ultrasonography (USG), contrast-enhanced abdominal computed tomography (CT), and magnetic resonance cholangio-pancreatography (MRCP) are favored diagnostic modalities with hemogram and biochemical indicators.

**Corresponding Author**: Burak Akın, burakakin3232@hotmail.com

This work is licensed under a Creative Commons Attribution 4.0 International License.

The clinical presentation ranges from a mild version that exhibits a prompt response to medical intervention, to a severe form characterized by systemic manifestations, sepsis, and multi-organ failure. The majority of patients exhibit a modest trajectory, leading to prompt clinical amelioration due to the implementation of moderate fluid resuscitation, pain and nausea treatment, and early initiation of oral feeding. In its severe form, which constitutes 20-30% of the patient group, hospital mortality rates can reach approximately 15%.[10] Infected necrosis affects mortality considerably, and mortality is 35.2% in cases with organ failure and infected necrosis, while mortality is at a lower level of 1.4% if there is infected necrosis without organ failure.[11]

The aim of early evaluation, diagnosis and treatment is to minimize complications and prevent morbidity and mortality. Today, although various diagnostic tests and imaging techniques are applied to direct the diagnosis, some delays can still be observed in the diagnosis and treatment of AP patients. Therefore, there is a constant need for studies on high-sensitivity biochemical biomarkers and imaging methods that can more rapidly and specifically evaluate the pathogenesis, diagnosis and prognosis of the disease in AP.

In this study, we aimed to contribute to the literature by evaluating the hospitalization and mortality status of acute pancreatitis cases, along with laboratory and imaging results.

## METHODS

The study was carried out with the permission of İstanbul Prof. Dr. Cemil Taşcıoğlu City Hospital Clinical Researches Ethics Committee (Date: 08.03.2021, Decision No: 105) All procedures were carried out in accordance with the ethical rules and the principles of the Declaration of Helsinki.

### Study Design and Population

The research encompassed a population of 225 individuals (116 females, 109 males) with a mean age of 54.10±19.07 years and a range of 18 to 94 years. These participants were diagnosed with acute pancreatitis in the emergency department during the period from 1 September 2020 to 1 March 2021. The inclusion criteria for this study required participants to be above the age of eighteen. The hospital where the study was conducted is a tertiary education and research hospital, and all records of patients' anamnesis, physical examination, laboratory and imaging reports are available in the electronic data system.

The diagnosis of acute pancreatitis was established based on the fulfillment of two affirmative criteria: the presence of abdominal pain that aligns with the symptoms associated with the disease, biochemical evidence indicating the existence of pancreatitis, and the identification of distinctive observations on abdominal imaging. As the biochemical proof of AP, a serum amylase and/or lipase three times or more above the normal value was accepted as diagnostic. Patients over the age of 18 who met these criteria, who did not have the diagnoses in the exclusion criteria and whose data in the hospital registry system were complete, were included in the study.

Patients with trauma, salivary gland disease, non-pancreatic infection, isolated cholecystitis, isolated choledoclithiasis, chronic pancreatitis, gastroenteritis, inflammatory bowel disease, obstructive bowel disease, celiac disease, pheochromocytoma, sarcoidosis, macroamylasemia, macrolipazemia, renal failure, malignancy clinic or history and under the age of 18 were excluded from the study. In addition, cases with missing data in the data recording system were not included in the study.

Age, gender, white blood cell (WBC), lymphocyte (LYM), neutrophil (NEU), urea, aspartate aminotransferase (AST), alanine aminotransferase (ALT), gamma glutamyl transferase (GGT), lactate dehydrogenase (LDH) were included in the patient case forms. Amylase, lipase and C-reactive protein (CRP) values were recorded. The presence of USG, CT, and MRCP examinations of the patients were recorded. The findings of the patients who underwent USG were evaluated in five groups: Gallbladder wall thickness increase (GWT), Gallstone (GS), intrahepatic bile duct dilatation (IBD), pancreatic edema (PE), peripancreatic fluid (PPF). CT and MRCP findings were evaluated in six groups by evaluating pancreatic density increase/heterogeneity (PDI) in addition to the findings on USG. Hospitalization status and duration and mortality status of the patients were also recorded. The patients were evaluated by dividing them into two groups according to their hospitalization and mortality status.

### Laboratory Design

Blood samples were collected from patients who were hospitalized to the emergency room with a preliminary diagnosis of AP in order to conduct hemogram, biochemistry, and C-reactive protein tests. The study examined the parameters measured upon admission to the emergency department, which provided a range of 45 to 90 minutes.

The hemogram was assessed utilizing a Beckman Coulter Automated CBC Analyzer, manufactured by Beckman Coulter, Inc. in Fullerton, CA, USA. The blood samples were subjected to biochemical analysis using the Cobas 6000 instrument (specifically, the C6000-Core module from the Cobas c-501 series, manufactured by Hitachi and distributed by Roche, USA).

### Statistical Analysis

The data acquired from the study were analyzed using the SPSS 20 software package developed by SPSS Inc., based in Chicago, IL, USA. The investigation of the normal distributions of the variables involved the utilization of the Kolmogorov-Smirnov test. The descriptive statistics were reported in the form of mean ± standard deviation or median (minimum-maximum) for continuous variables, and as the count and percentage (%) for nominal variables. The Mann-Whitney U test was employed to analyze the disparities between groups due to the non-normal distribution of the variables. The use of chi-square analysis was employed to investigate the associations among groups of nominal variables. Statistical significance was determined by considering values below the significance level of 0.05 throughout the interpretation of the results.

## RESULTS

The study included a total of 225 cases of acute pancreatitis, with a mean age of 54.10±19.07 years. Among these cases, 116 individuals (51.6%) were identified as women. Out of the total number of patients, 169 individuals (75.1%) were hospitalized, while 56 patients (24.9%) were released from the emergency department. There was no significant association found between age (p=0.362) and gender (p=0.308) with hospitalization. The mean duration of hospitalized patients was 4.75±3.83 days. In the hospitalization group, amylase was 964±1101.1 U/L (p<0.001), lipase was 1902.34±2199.99 U/L (p<0.001) and there was a significant increase in hospitalized patients. The GGT value was 232.98±297.24 U/L in the hospitalization group and was significant (p=0.021). Total bilirubin value was significantly higher in the hospitalization group with 2.16±3.02 mg/dl (p=0.018). Other laboratory parameters were not associated with hospitalizations (Table 1).

The mean age of the survival group was 53.24±18.71 years, and 81.00±6.80 years in the mortality group (p<0.001). Seven (3.1%) cases resulted in mortality. Of the cases that resulted in mortality, 6 (2.7%) were female (p=0.070). The duration of hospitalization was found to be 7.43±2.64 days, with a statistically significant increase observed in the mortality group (p=0.002). Although there was no observed association between amylase values and death, it was found that lipase levels were considerably elevated in the group who had mortality, with an average of 3474.71±3013.69 U/L (p=0.046). In addition, high urea was also evaluated to be associated with mortality (p=0.019). All hemogram and biochemistry parameters evaluated except urea and lipase were not associated with mortality (Table 2).

**Table 1.** The relationship of hospitalization with age, gender and laboratory parameters

| | All Patients n(%) | Hospitalization (-) n(%) | Hospitalization (+) n(%) | p value |
|---|---|---|---|---|
| Gender | | | | 0.308 |
| Female | 116(51.6) | 31(13.8) | 85(37.8) | |
| Male | 109(48.4) | 25(11.1) | 84(37.3) | |
| Total | 225(100) | 56(24.9) | 169(75.1) | |
| | **Mean ±SD** | **Mean ±SD** | **Mean ±SD** | |
| Age (year) | 54.10±19.07 | 52.07±18.31 | 54.77±19.32 | 0.362 |
| Hospitalization Time (day) | 3.59±3.90 | | 4.75±3.83 | |
| Laboratory Findings | | | | |
| WBC($10^3$/µl) | 11.12±4.77 | 10.46±4.59 | 11.34±4.83 | 0.156 |
| NEU($10^3$/µl) | 8.42±4.55 | 7.52±4.15 | 8.72±4.66 | 0.053 |
| LYM($10^3$/µl) | 1.81±1.09 | 2.01±1.49 | 1.74±0.92 | 0.379 |
| Urea (mg/dl) | 49.36±55.28 | 47.71±47.78 | 49.90±57.67 | 0.764 |
| ALT(U/L) | 153.16±296.90 | 117.73±170.51 | 164.90±327.81 | 0.061 |
| AST(U/L) | 172.05±453.86 | 123.98±188.98 | 187.98±511.79 | 0.069 |
| GGT (U/L) | 218.51±292.09 | 174.86±273.86 | 232.98±297.24 | 0.021 |
| LDH (U/L) | 441.37±928.27 | 377.71±365.78 | 462.46±1050.38 | 0.088 |
| Amylase (U/L) | 855.74±1084.76 | 529.03±972.06 | 964±1101.01 | <0.001 |
| Lipase (U/L) | 1700.02±2138.51 | 1089.44±2095.69 | 1902.34±2119.99 | <0.001 |
| CRP (mg/dl) | 41.37±65.84 | 27.79±51.24 | 45.87±69.56 | 0.039 |
| Direct Bilirubin (mg/dl) | 0.96±1.59 | 0.72±1.29 | 1.04±1.67 | 0.218 |
| Total Bilirubin (mg/dl) | 2.02±2.89 | 1.59±2.43 | 2.16±3.02 | 0.018 |
| Sodium (mEq/L) | 135.77±4.92 | 135.11±4.89 | 135.99±4.92 | 0.278 |
| Potassium (mEq/L) | 4.28±0.66 | 4.26±0.65 | 4.28±0.66 | 0.659 |

SD: standard deviation, WBC: White Blood Cell, NEU: Neutrophil, LYM: Lymphocyte, ALT: Alanine Aminotransferase, AST: Aspartate Aminotransferase, GGT: gamma glutamyl transferase, LDH: Lactate dehydrogenase, CRP: C-Reactive Protein, p:Statistical significance (<0.05)

**Table 2.** Relationship of mortality with age, gender and laboratory parameters

| | Mortality (-) n(%) | Mortality (+) n(%) | p value |
|---|---|---|---|
| Gender | | | 0.070 |
| Female | 110(48.9) | 6(2.7) | |
| Male | 108(48) | 1(0.4) | |
| Total | 218(96.9) | 7(3.1) | |
| | **Mean ±SD** | **Mean ±SD** | |
| Age (year) | 53.24±18.71 | 81.00±6.80 | <0.001 |
| HosiptalizationTime (day) | 3.47±3.88 | 7.43±2.64 | 0.002 |
| **Laboratory Findings** | | | |
| WBC($10^3$/µl) | 11.07±4.79 | 12.56±4.36 | 0.286 |
| NEU($10^3$/µl) | 8.37±4.55 | 10.28±4.84 | 0.217 |
| LYM($10^3$/µl) | 1.81±1.11 | 1.68±0.80 | 0.904 |
| Urea (mg/dl) | 46.90±50.01 | 125.88±128.72 | 0.019 |
| ALT(U/L) | 155.44±300.95 | 82.14±95.92 | 0.481 |
| AST(U/L) | 171.50±459.37 | 189.14±240.93 | 0.662 |
| GGT (U/L) | 221.90±295.03 | 113.00±153.76 | 0.181 |
| LDH (U/L) | 438.22±940.21 | 539.28±432.42 | 0.156 |
| Amylase (U/L) | 849.71±1092.44 | 1043.71±851.82 | 0.264 |
| Lipase (U/L) | 1643.04±2089.20 | 3474.71±3013.69 | 0.046 |
| CRP (mg/dl) | 39.66±61.94 | 94.56±140.27 | 0.330 |
| Direct Bilirubin (mg/dl) | 0.98±1.61 | 0.42±0.31 | 0.908 |
| Total Bilirubin (mg/dl) | 2.05±2.93 | 1.12±0.66 | 0.848 |
| Sodium (mEq/L) | 135.72±4.95 | 137.43±3.78 | 0.556 |
| Potassium (mEq/L) | 4.27±0.66 | 4.47±0.55 | 0.115 |

SD: standard deviation, WBC: White Blood Cell, NEU: Neutrophil, LYM: Lymphocyte, ALT: Alanine Aminotransferase, AST: Aspartate Aminotransferase, GGT: gamma glutamyl transferase, LDH: Lactate dehydrogenase, CRP: C-Reactive Protein, p:Statistical significance (<0.05)

In the evaluation of hospitalization and mortality with imaging results, USG findings were not associated with hospitalization, but pancreatic edema was found to be associated with mortality (p=0.012). CT and MRCP findings were not associated with the mortality of the cases. Gallbladder wall thickness increase (p=0.016) and increase in pancreatic density and heterogeneity (p=0.038) hospitalizations were associated as CT findings. In addition, the presence of intrahapatic bill duct dilatation (p=0.002), pancreatic edema (p=0.045) and peripancreatic fluid (p=0.009) in MRCP was also evaluated as associated with hospitalization (Table 3).

Figure 1 displays the analysis of mortality through the Receiver Operating Characteristic (ROC) curve. Based on the findings of this analysis, it has been determined that there are optimal cut-off values for amylase and lipase in predicting the development of mortality. The amylase test demonstrated a sensitivity of 47.9% and a specificity of 44.5%, with an area under the curve (AUC) of 0.624 (95% confidence interval: 0.414-0.834, p=0.264). On the other hand, the lipase test showed a sensitivity of 71.4% and a specificity of 69.3%, with an AUC of 0.722 (95% confidence interval: 0.529-0.915, p=0.046). These results were observed when the values exceeded 45%.

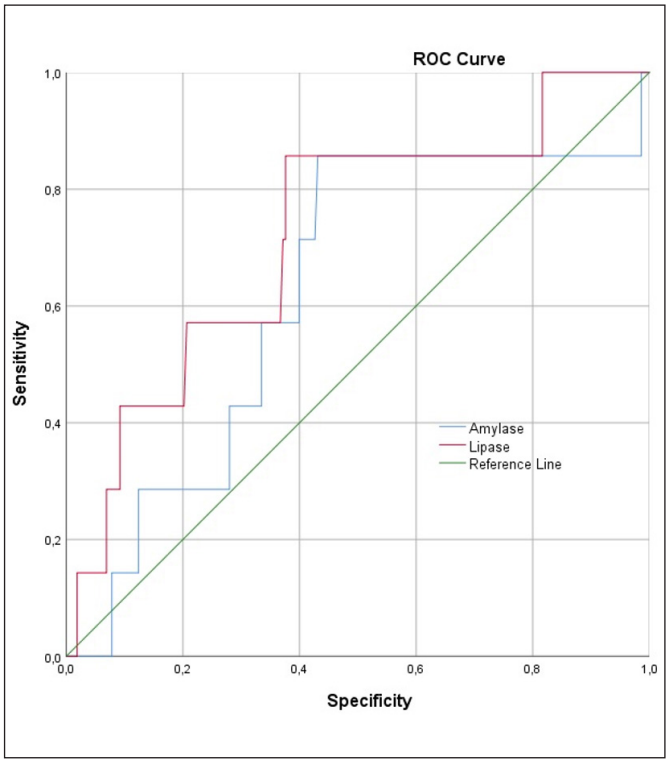


**Figure 1.** ROC Curve in terms of the relationship of amylase and lipase values with mortality

**Table 3.** Relation of hospitalization and mortality presence with gender and imaging findings

| | | H(-) n(%) | H(+) n(%) | Total n(%) | p value | M(-) n(%) | M(+) n(%) | Total n(%) | p value |
|---|---|---|---|---|---|---|---|---|---|
| **Ultrasonography** | | | | | | | | | |
| USG | No | 14 (6.2) | 14 (6.2) | 28 (12.4) | 0.002 | 28 (12.4) | 0 (0) | 28 (12.4) | 0.389 |
| | Yes | 42 (18.7) | 155 (68.9) | 197 (87.6) | | 190 (84.4) | 7 (3.1) | 197 (87.6) | |
| GWT | No | 52 (23.1) | 145 (64.4) | 197 (87.6) | 0.122 | 191 (84.9) | 6 (2.7) | 197 (87.6) | 0.611 |
| | Yes | 4 (1.8) | 24 (10.7) | 28 (12.4) | | 27 (12) | 1 (0.4) | 28 (12.4) | |
| GS | No | 42 (18.7) | 105 (46.7) | 147 (65.3) | 0.054 | 143 (63.6) | 4 (1.8) | 147 (65.3) | 0.460 |
| | Yes | 14 (6.2) | 64 (28.4) | 78 (34.7) | | 75 (33.3) | 3 (1.3) | 78 (34.7) | |
| IBD | No | 48 (21.3) | 126 (56) | 174 (77.3) | 0.058 | 169 (75.1) | 5 (2.2) | 174 (77.3) | 0.497 |
| | Yes | 8 (3.6) | 43 (19.1) | 51 (22.7) | | 49 (21.8) | 2 (0.9) | 51 (22.7) | |
| PE | No | 55 (24.4) | 164 (72.9) | 219 (97.3) | 0.535 | 214 (95.1) | 5 (2.2) | 219 (97.3) | 0.012 |
| | Yes | 1 (0.4) | 5 (2.2) | 6 (2.7) | | 4 (1.8) | 2 (0.9) | 6 (2.7) | |
| PPF | No | 55 (24.4) | 159 (70.7) | 214 (95.1) | 0.192 | 209 (92.9) | 5 (2.2) | 214 (95.1) | 0.040 |
| | Yes | 1 (0.4) | 10 (4.4) | 11 (4.9) | | 9 (4) | 2 (0.9) | 11 (4.9) | |
| **Computerized tomography** | | | | | | | | | |
| CT | No | 42 (18.7) | 83 (36.9) | 125 (55.6) | 0.001 | 120 (53.3) | 5 (2.2) | 125 (55.6) | 0.324 |
| | Yes | 14 (6.2) | 86 (38.2) | 100 (44.4) | | 98 (43.6) | 2 (0.9) | 100 (44.4) | |
| GWT | No | 56 (24.9) | 155 (68.9) | 211 (93.8) | 0.016 | 204 (90.7) | 7 (3.1) | 211 (93.8) | 0.634 |
| | Yes | 0 (0) | 14 (6.2) | 14 (6.2) | | 14 (6.2) | 0 (0) | 14 (6.2) | |
| GS | No | 54 (24) | 157 (69.8) | 211 (93.8) | 0.276 | 205 (91.1) | 6 (2.7) | 211 (93.8) | 0.366 |
| | Yes | 2 (0.9) | 12 (5.3) | 14 (6.2) | | 13 (5.8) | 1 (0.4) | 14 (6.2) | |
| IBD | No | 54 (24) | 155 (68.9) | 209 (92.9) | 0.190 | 202 (89.8) | 7 (3.1) | 209 (92.9) | 0.592 |
| | Yes | 2 (0.9) | 14 (6.2) | 16 (7.1) | | 16 (7.1) | 0 (0) | 16 (7.1) | |
| PE | No | 52 (23.1) | 155 (68.9) | 207 (92) | 0.521 | 201 (89.3) | 6 (2.7) | 207 (92) | 0.447 |
| | Yes | 4 (1.8) | 14 (6.2) | 18 (8) | | 17/7.6) | 1 (0.4) | 18 (8) | |
| PPF | No | 54 (24) | 152 (67.6) | 206 (91.6) | 0.103 | 199 (88.4) | 7 (3.1) | 206 (91.6) | 0.535 |
| | Yes | 2 (0.9) | 17 (7.6) | 19 (8.4) | | 19 (8.4) | 0 (0) | 19 (8.4) | |
| PDI | No | 51 (22.7) | 135 (60) | 186 (82.7) | 0.038 | 180 (80) | 6 (2.7) | 186 (82.7) | 0.651 |
| | Yes | 5 (2.2) | 34 (15.1) | 39 (17.3) | | 38 (16.9) | 1 (0.4) | 39 (17.3) | |
| **Magnetic resonance imaging** | | | | | | | | | |
| MRCP | No | 48 (21.3) | 106 (47.1) | 154 (68.4) | 0.001 | 148 (65.8) | 6 (2.7) | 154 (68.4) | 0.293 |
| | Yes | 8 (3.6) | 63 (28) | 71 (31.6) | | 70 (31.1) | 1 (0.4) | 71 (31.6) | |
| GWT | No | 53 (23.6) | 154 (68.4) | 207 (92) | 0.300 | 200 (88.9) | 7 (3.1) | 207 (92) | 0.553 |
| | Yes | 3 (1.3) | 15 (6.7) | 18 (8) | | 18 (8) | 0 (0) | 18 (8) | |
| GS | No | 51 (22.7) | 139 (61.8) | 190 (84.4) | 0.082 | 183 (81.3) | 7 (3.1) | 190 (84.4) | 0.301 |
| | Yes | 5 (2.2) | 30 (13.3) | 35 (15.6) | | 35 (15.6) | 0 (0) | 35 (15.6) | |
| IBD | No | 53 (23.6) | 131 (58.2) | 184 (81.8) | 0.002 | 178 (79.1) | 6 (2.7) | 184 (81.8) | 0.626 |
| | Yes | 3 (1.3) | 38 (16.9) | 41 (18.2) | | 40 (17.8) | 1 (0.4) | 41 (18.2) | |
| PE | No | 55 (24.4) | 153 (68) | 208 (92.4) | 0.045 | 201 (89.3) | 7 (3.1) | 208 (92.4) | 0.573 |
| | Yes | 1 (0.4) | 16 (7.1) | 17 (7.6) | | 17 (7.6) | 0 (0) | 17 (7.6) | |
| PPF | No | 56 (24.9) | 153 (68) | 209 (92.9) | 0.009 | 202 (89.8) | 7 (3.1) | 209 (92.9) | 0.592 |
| | Yes | 0 (0) | 16 (7.1) | 16 (7.1) | | 16 (7.1) | 0 (0) | 16 (7.1) | |
| PDI | No | 55 (24.4) | 155 (68.9) | 210 (93.3) | 0.075 | 204 (90.7) | 6 (2.7) | 210 (93.3) | 0.387 |
| | Yes | 1 (0.4) | 14 (6.2) | 15 (6.7) | | 14 (6.2) | 1 (0.4) | 15 (6.7) | |
| Total | | 56 (24.9) | 169 (75.1) | 225 (100) | | 218 (96.9) | 7 (3.1) | 225 (100) | |

H: Hospitalization, M: Mortality, GWT: Gallbladder Wall Thickness Increase, GS: Gallstone, IBD: Intrahepatic Bile Duct Dilatation, PE: Pancreatic Edema, PDI: Pancreatic Density Increase/heterogeneity, PPF: Peripancreatic Fluid, USG: Ultrasonography, CT: Computerized Tomography, MRCP: Magnetic Resonance Cholangiopancreatography, p:Statistical significance (<0.05)

## DISCUSSION

In AP, which is an inflammatory condition that can cause even multi-systemic organ failure with a severe mortality rate, laboratory analysis is essential in addition to anamnesis and physical examination for appropriate evaluation.[12] It is also very important to make the diagnosis of AP quickly, to start the treatment early and to prevent the complications that may develop. Although many different biomarkers are used in the diagnosis of AP, there is still no biomarker with high sensitivity and specificity. There are also no clear ideas about how the results will affect the prognosis. In addition to basic laboratory diagnostic parameters, imaging methods are also very important in terms of both diagnosis and treatment. We aimed to evaluate whether some laboratory parameters, especially amylase and lipase, and imaging options applied to the patients could give an idea about the hospitalization and mortality status of the patients.

Although the mean age in our study was similar to other studies in the literature, it had a wide age range of 18-94 years. The wide age range is important in terms of considering AP in the differential diagnosis of all ages. While some studies have reported that acute pancreatitis is more common in women, some studies have found that AP is more common in men.[13-15]

Despite the absence of a statistically significant disparity, our investigation revealed that the proportion of female patients was 51.6%, indicating a higher representation compared to other groups. The study conducted by Shen et al. examined the impact of gender on mortality in a sample of 13,110 AP patients. The results indicated a statistically significant association between gender and mortality, with men exhibiting greater mortality rates.[16] In our study, there was a significant increase in mortality with age, similar to other studies. Although no statistically significant correlation was observed between mortality and gender, 6 (2.7%) of 7 (3.1%) cases resulted in mortality were women.

According to the results of a study, the average length of hospital stay for AP-related hospitalizations was 6.4 days in 1997 and 4.7 days in 2003.[3] In a retrospective observational study of 232 patients presenting with the first mild acute pancreatitis attack, Francisco et al.[17] examined the factors associated with long hospital stay in mild acute pancreatitis. In this study, the mean hospital stay was 8 days.

In our study, the average length of stay was 4.75 days only in patients hospitalized, while it was 3.6 days among all pancreatitis patients. The observed reduction in length of stay can likely be attributed to advancements in the comprehension of the pathophysiology of AP, enhanced identification of complications at an earlier stage, superior management strategies for these complications, and a heightened realization of the imperative to mitigate healthcare expenditures. White blood cells (WBCs) play a crucial role in the initiation and regulation of the inflammatory response. Hematopoietic stem cells, which are multipotent cells located in the bone marrow, serve as the source and origin of all WBCs. White blood cells play many roles in the promotion and regulation of inflammation.[18] According to reports, there exists a correlation between elevated leukocyte count, specifically a rise in WBC count, and mortality. Especially the fact that pancreatic necrosis leads to the development of systemic inflammatory response syndrome emphasizes the importance of leukocytes and WBC in pancreatitis.[19] Contrary to this information, in our study, no relationship was found between WBC and hospitalization and mortality.

As it is known, AP is an inflammatory process and in a study conducted with CRP, one of the markers showing inflammation, it was stated that CRP values of >190 mg/dl could indicate the severity of acute pancreatitis.[20] In another study, Sternby et al.[21] mentioned that CRP can be used to differentiate between moderate and severe pancreatitis. In our study, it was determined that high CRP value was associated with hospitalization in AP cases.

In the research of Faisst et al.,[22] high BUN values at admission and an increase in BUN values during the course of the disease in patients with acute necrotizing pancreatitis were found to be associated with long stays in the ICU (≥14 days), and mortality was significantly increased in these patients. Renal failure was also found to be an important risk factor prolonging hospital stay. Francisco et al.[23] also reported in their study that urea was associated with hospitalization and long stay. Although urea was not associated with hospitalization in our study, it was observed that it had a significant effect on mortality.

The sensitivity of lipase level surpasses that of amylase level. Amylase is synthesized in the salivary glands and can be found at normal levels in individuals with recurrent alcoholic pancreatitis.[23] Pancreatitis can be diagnosed when the levels of lipase or amylase exceed three times the upper limit of the normal range.[24] According to a report, there is no observed correlation between blood amylase and lipase levels and the clinical severity of acute pancreatitis.[25] Although there are studies evaluating the relationship of amylase and lipase with the severity of the disease, in our study, in which hospitalization and mortality were evaluated, both amylase velipase is associated with hospitalization. This can be attributed to the fact that these two parameters are already in the diagnostic criteria. However, in addition to hospitalization, lipase was also associated with mortality in our study.

Imaging methods are very important in AP, both in the diagnosis, in the determination of complications and in the treatment process. Patients' need for abdominal imaging and procedures such as CT and MRCP have been associated with long hospital stays.[17] In the study, USG was evaluated in 197 (87.6%) of our cases, CT in 100 (44.4%) and MRCP in 71 (31.6%) of our cases. While USG, CT and MRCP examination were not associated with mortality in our study, all three were found to be associated with hospitalization. According to the study conducted by Greenberg et al.,[26] it is strongly recommended, with high levels of evidence, that USG should be conducted as the initial diagnostic procedure for all patients presenting with acute pancreatitis. The primary objective of this procedure is to identify the presence of gallstones and/or stones in the common bile duct, as well as to assess the condition of the biliary tract. In the research conducted by Karaca and Oktay,[27] abdominal USG was conducted on all individuals in the emergency department. The USG findings indicated pancreatitis in 30 patients (25.9%), while it did not indicate pancreatitis in 61 patients (52.6%). Additionally, 25 patients (21%) had

inconclusive USG results regarding pancreatitis. In our study, USG findings were not associated with hospitalization, but the presence of pancreatic edema on USG was associated with mortality. We attribute the absence of this relationship in CT and MRCP to the fact that advanced edema can be seen on USG and this is associated with an unfavorable prognosis. The efficacy of abdominal USG in pancreatic imaging may be constrained by factors such as the presence of intestinal gas and obesity. Indeed, it can be postulated that the solicitation of abdominal ultrasonography in the emergency department has the potential to impede patient care in this specific cohort. Nevertheless, it is our contention that abdominal USG ought to be administered to all individuals presenting with acute pancreatitis in the emergency department. This is because it plays a significant role in the care of patients with acute pancreatitis and aids in ruling out surgical or alternative etiologies of acute abdominal pain. Contrast-enhanced CT is the gold standard for the diagnosis and evaluation of acute pancreatitis.[28] CT defines anatomical structures better and can reveal complications such as pancreatic inflammation and necrosis. CT is also helpful in determining clinical severity and prognosis, with a diagnostic value of 75–90% for acute pancreatitis.[29,30] MRI and MRCP are good choices for demonstrating pancreatobiliary anomalies because they take multislice images. MRCP is important in elucidating the biliary etiology of pancreatitis. The ideal imaging time for MRCP is when pancreatic edema regresses and the acute attack subsides.[31]

We did not find any detailed study in which CT and MRCP findings were associated with mortality. In our study, we found that none of the findings were associated with mortality in the evaluation made according to the hospitalization and mortality status of the patients. However, increase in gallbladder wall thickness and increase in pancreatic density/heterogeneity in CT, enlargement of intrahepatic bile ducts, pancreatic edema and peripancreatic fluid in MRCP were associated with hospitalization.

### Study Limitations

The study had some limitations. The most important of these limitations is related to the retrospective and single-center planning of the study. The information of the patients was obtained from the hospital electronic database and the files in the hospital archive. The exclusion of some of the patients from the study due to errors and deficiencies in the recording of these data may also be a limitation in reducing the effectiveness of the study.

## CONCLUSION

Acute pancreatitis is a frequent cause of admission to emergency departments and is still an important problem in morbidity and mortality. Correct diagnosis and early treatment are very important. Being able to use laboratory and imaging methods correctly and being able to make an evaluation in the direction of predicting prognosis and mortality contributes positively to the process. Although there are many studies in terms of clinical severity, laboratory, imaging and prognosis, studies are limited in terms of the effectiveness of laboratory and imaging results in hospitalization and mortality. Amylase, lipase, GGT and Significantly elevated CRP results were observed in patients who were hospitalized. In terms of mortality, especially high urea and lipase values should be a warning in acute pancreatitis. In imaging methods, some findings are significant in hospitalized patients, but no CT or MRCP finding that will give an idea about mortality has been detected. However, the presence of edema and peripancreatic fluid in the pancreas on USG should be a warning for mortality. Prospective studies are needed on the clinical and prognostic effects of laboratory and imaging parameters in AP.

## ETHICAL DECLARATIONS

**Ethics Committee Approval:** The study was carried out with the permission of İstanbul Prof. Dr. Cemil Taşcıoğlu City Hospital Clinical Researches Ethics Committee (Date: 08.03.2021, Decision No: 105)

**Informed Consent:** Because the study was designed retrospectively, no written informed consent form was obtained from patients.

**Referee Evaluation Process:** Externally peer-reviewed.

**Conflict of Interest Statement:** The authors have no conflicts of interest to declare.

**Financial Disclosure:** The authors declared that this study has received no financial support.

**Author Contributions:** All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

## REFERENCES

1. Akshintala VS, Kamal A, Singh VK. Uncomplicated acute pancreatitis: evidenced-based management decisions. *Gastrointest Endosc Clin N Am*. 2018;28(4):425-438.
2. Fagenholz PJ, Castillo CF, Harris NS, Pelletier AJ, Camargo CA Jr. Increasing United States hospital admissions for acute pancreatitis, 1988-2003. *Ann Epidemiol*. 2007;17(7):491-497.
3. Peery AF, Crockett SD, Barritt AS, et al. Burden of gastrointestinal, liver, and pancreatic diseases in the United States. *Gastroenterology*. 2015;149(7):1731-1741.e3.

4. Tan JW, Zhang XQ, Geng CM, Peng LL. Development of the national early warning score-calcium model for predicting adverse outcomes in patients with acute pancreatitis. *J Emerg Nurs.* 2020;46(2):171-179.
5. Gullo L, Migliori M, Oláh A, et al. Acute pancreatitis in five European countries: etiology and mortality. *Pancreas.* 2002;24(3):223-227.
6. Malka D, Rosa-Hézode I. Comment faire le diagnostic positif et étiologique de pancréatite aiguë? [Positive and etiological diagnosis of acute pancreatitis]. *Gastroenterol Clin Biol.* 2001;25(1 Suppl):1S153-1S168.
7. Banks PA, Bollen TL, Dervenis C, et al. Classification of acute pancreatitis--2012: revision of the Atlanta classification and definitions by international consensus. *Gut.* 2013;62(1):102-111.
8. Carroll JK, Herrick B, Gipson T, Lee SP. Acute pancreatitis: diagnosis, prognosis, and treatment. *Am Fam Physician.* 2007;75(10):1513-1520.
9. Maher MM, Lucey BC, Gervais DA, Mueller PR. Acute pancreatitis: the role of imaging and interventional radiology. *Cardiovasc Intervent Radiol.* 2004;27(3):208-225.
10. van Santvoort HC, Bakker OJ, Bollen TL, et al. A conservative and minimally invasive approach to necrotizing pancreatitis improves outcome. *Gastroenterology.* 2011;141(4):1254-1263.
11. Werge M, Novovic S, Schmidt PN, Gluud LL. Infection increases mortality in necrotizing pancreatitis: A systematic review and meta-analysis. *Pancreatology.* 2016;16(5):698-707.
12. Weiss FU, Laemmerhirt F, Lerch MM. Acute pancreatitis: genetic risk and clinical implications. *J Clin Med.* 2021;10(2):190.
13. Coşkun BN, Tandoğan G, Eroğlu A, et al. Akut pankreatit tanılı hastaların etyolojik ve prognostik faktörlerinin retrospektif incelenmesi. Uludağ Tıp Derg. 2012;38(2):67-73.
14. Demiral G, Yener O, Aksoy F, et al. Retrospective analysis of our acute pancreatitis patients. *Göztepe Med J.* 2011;26(1):4-9.
15. Ertas H, Duran C, Keskin M, Güney I. Examining the clinical and laboratory characteristics of patients diagnosed with acute pancreatitis. *Aegean J Med Sci.* 2018;3:97-102.
16. Shen HN, Wang WC, Lu CL, Li CY. Effects of gender on severity, management and outcome in acute biliary pancreatitis. *PLoS One.* 2013;8(2):e57504.
17. Francisco M, Valentín F, Cubiella J, Fernández-Seara J. Factors related to length of hospital admission in mild interstitial acute pancreatitis. *Rev Esp Enferm Dig.* 2013;105(2):84-92.
18. De Campos T, Cerqueira C, Kuryura L, et al. Morbimortality indicators in severe acute pancreatitis. *JOP.* 2008;9(6):690-697.
19. Mare TA, Treacher DF, Shankar-Hari M, et al. The diagnostic and prognostic significance of monitoring blood levels of immature neutrophils in patients with systemic inflammation. *Crit Care.* 2015;19(1):57.
20. Stirling AD, Moran NR, Kelly ME, Ridgway PF, Conlon KC. The predictive value of C-reactive protein (CRP) in acute pancreatitis - is interval change in CRP an additional indicator of severity?. *HPB (Oxford).* 2017;19(10):874-880.
21. Sternby H, Hartman H, Johansen D, Thorlacius H, Regnér S. IL-6 and CRP are superior in early differentiation between mild and non-mild acute pancreatitis. *Pancreatology.* 2017;17(4):550-554.
22. Faisst M, Wellner UF, Utzolino S, Hopt UT, Keck T. Elevated blood urea nitrogen is an independent risk factor of prolonged intensive care unit stay due to acute necrotizing pancreatitis. *J Crit Care.* 2010;25(1):105-111.
23. Smotkin J, Tenner S. Laboratory diagnostic tests in acute pancreatitis. *J Clin Gastroenterol.* 2002;34(4):459-462.
24. American Gastroenterological Association (AGA) Institute on "Management of Acute Pancreatits" Clinical Practice and Economics Committee; AGA Institute Governing Board. AGA Institute medical position statement on acute pancreatitis. *Gastroenterology.* 2007;132(5):2019-2021.
25. Vissers RJ, Abu-Laban RB. Acute and chronic pancreatitis. In: Tintinalli JE, Kelen GD, Stapczynski JS (eds.), Emergency medicine. A comprehensive study guide. 5th edition, New York: McGraw-Hill, 2000:pp.588-592.
26. Greenberg JA, Hsu J, Bawazeer M, et al. Clinical practice guideline: management of acute pancreatitis. *Can J Surg.* 2016;59(2):128-140.
27. Karaca E, Oktay C. The effect of prognostic criteria on the outcome of patients with nontraumatic acute pancreatitis. *Turk J Emerg Med.* 2008;8:18-25.
28. Sargent S. Pathophysiology, diagnosis and management of acute pancreatitis. *Br J Nurs.* 2006;15(18):999-1005.
29. Kim YS, Lee BS, Kim SH, Seong JK, Jeong HY, Lee HY. Is there correlation between pancreatic enzyme and radiological severity in acute pancreatitis?. *World J Gastroenterol.* 2008;14(15):2401-2405.
30. Mitchell RM, Byrne MF, Baillie J. Pancreatitis. *Lancet*. 2003;361(9367):1447-1455.
31. Abu-El-Haija M, Kumar S, Quiros JA, et al. Management of acute pancreatitis in the pediatric population: a clinical report from the North American Society for Pediatric Gastroenterology, Hepatology and Nutrition Pancreas Committee. *J Pediatr Gastroenterol Nutr.* 2018;66(1):159-176.